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Periodontal risk

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ABSTRACT

Periodontal diseases are chronic inflammatory conditions primarily initiated by microbial biofilms. However, not all individuals with similar plaque accumulation develop periodontitis, indicating the influence of various risk factors. This review aims to summarize the evidence-based general and individual risk factors, indicators, and predictors associated with periodontal disease. General risk factors include microbial pathogens, poor oral hygiene, smoking, and diabetes mellitus. Individual risk factors encompass non-modifiable elements such as genetic predisposition, age, gender, socioeconomic status, and psychological stress. Additionally, conditions like HIV/AIDS, osteoporosis, and irregular dental visits are identified as risk indicators, while clinical findings such as bleeding on probing and history of periodontitis serve as risk predictors. Understanding and evaluating these factors are crucial for preventive strategies, disease management, and improving periodontal prognosis.

Keywords: Periodontal disease, risk factors, risk indicators, risk predictors

STRENGTH OF EVIDENCE FOR RISK DEFINITION AND RISK FACTORS

The word "risk" originates from French, and its root word is "riziko." The dictionary definition of "riziko" is the danger of suffering loss.[1]

In health research, risk is defined as the likelihood of a person developing a specific disease over time. Risk is the probability of an event occurring. In epidemiology, it is often used to indicate the probability of a specific event occurring.[2] In chronic diseases and infections, the same outcome is not always achieved; there are very few situations that constitute sufficient cause.[3]

The impact of each scientific study on epidemiological evidence is determined not only by the quality of the study but also by the design and methods used, which determine what conclusions about causality can be drawn from the data obtained. In human health, a causal factor is defined as a factor whose presence increases the occurrence of a disease or condition.[4]

To establish causality, certain criteria must be met, such as the presence of the cause before the disease outcome (temporality) and biological plausibility.[5]

Potential risk factors can be identified through data obtained from epidemiological studies for use in subsequent advanced studies. These indicators then provide evidence that sheds light on how etiology and risk factors work at the cellular, genetic, and molecular levels.[6]

After reviewing the evidence, recommendations can be made for modifying risk factors for preventive measures in public health.[4]

One of the most complex issues in epidemiological studies is determining causal inferences. In 1971, Hill defined the criteria that must be met to see causal inferences:[7]

Strength of the Relationship

The stronger the relationship between a potential risk factor and the occurrence of a disease, the more valid the expected causal relationship. As the strength of the relationship increases, the likelihood of causation increases; estimated relative risk and relative risk confirm this strength.

Dose-Response Effect

If the frequency of the disease increases as the dose or level of exposure to a factor increases, this supports a causal relationship. An increase in the factor's presence and dose is expected to lead to an increase in the disease.

Temporal Consistency

It is important to determine whether exposure to the suspected factor occurred before the disease developed. This may be difficult to determine in diseases with a long latent period or where factors may change over time.

Consistency of Findings

Another criterion that strengthens causal interpretation is the obtaining of similar results in multiple studies examining a specific relationship.

Biological Plausibility

The expected relationship should be plausible when evaluated in light of existing biological knowledge. However, it should

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be noted that when data on the etiology of a particular disease are limited, this criterion may be more difficult to meet.

Specificity of the Relationship

If the disease is found to be associated with only one of the factors under investigation, or if the factors under investigation are associated with only one disease, the causal relationship is strengthened.

GENERAL RISK FACTORS

Periodontal disease is an infectious bacterial disease caused by dental plaque. The virulence of the main pathogens found in the subgingival biofilm can influence the etiology and pathogenesis of the disease. In the 1960s, people thought everyone was equally likely to get periodontal disease, but later studies using measurements like pocket depth, attachment loss, and gingival inflammation showed that only some people experience severe periodontitis, indicating that certain individuals are more at risk for the disease. This has led to the conclusion that some individuals are more susceptible to periodontal disease.[9]

Risk factors were defined by Beck[10] in the 1996 World Journal of Periodontology. They are defined as environmental, behavioral, or biological factors that, when present, directly increase the likelihood of disease onset, and when absent or eliminated, substantially reduce the likelihood of disease onset, as identified in longitudinal studies with a temporal sequence.

Behavioral, environmental, or biological factors that increase a person's likelihood of developing a disease are risk factors. These factors are links in the causal chain and are directly related to the occurrence of the disease. These factors are identified through long-term studies of individuals who have experienced the disease in question. Exposure to a risk factor may occur within a specific time frame, may occur multiple times at different times, or may be continuous over a long period of time. For a factor to be defined as a risk factor, exposure to the factor must occur before the onset of the disease.[11]

Pathogenic Bacteria and Adjuvants

Periodontitis is a chronic inflammatory disease that can cause progressive destruction of the tissues supporting the teeth if left untreated.[12] The accumulation of microbial plaque and the host's immune and inflammatory responses to enzymes such as nucleases, lipases, and proteases are the primary etiological factors of periodontal disease.[13] One of the most heterogeneous microbial communities found in humans is the oral microbiota, which ranks second after the gastrointestinal microbiota in terms of complexity and species diversity.[14] The complexity is due to the different environmental conditions in different regions of the oral cavity. Different environmental conditions (gingival sulcus, tongue, teeth, lips, hard and soft palate, buccal mucosa, keratinized gingiva, and non-keratinized gingiva) provide distinct habitats for microbial colonization. This allows microorganisms to obtain suitable environments for biofilm formation.[15]

The host's initial response to periodontal disease is an acute inflammation against supragingival and subgingival plaque, managed by components of the innate immune system such as epithelial cells, macrophages, neutrophils, fibroblasts, complement proteins, and neuropeptides. At this stage, cytokines such as interleukin-1β, interleukin-6, and tumor necrosis factor-α, produced by resident cells, increase the expression of adhesion molecules for neutrophils on the inner surface of blood vessels, cell migration to the infection site, and the synthesis of other pro-inflammatory cytokines.[16]

The elimination of the plaque contributes to the gradual resolution of inflammation and the reestablishment of individual homeostasis. The continued presence of the plaque triggers the activation of the acquired immune response. Antigens are processed and presented by lymphocytes, dendritic cells, and macrophages. These processes are regulated by acquired immune cytokines such as interferon (IFN)-γ and interleukin-2 and interleukin-4. Inflammation and cytokines disrupt the balance between osteogenesis and osteoclastogenesis in bone tissue, shifting it toward osteoclastogenesis and causing destruction.[17]

Axelsson et al.[18] conducted a study involving 375 participants over a 15-year period. All participants received oral hygiene education, and tartar removal and root planing were performed every two or three months for nine years, followed by twice a year for the next six years. The results indicated that no clinically detectable attachment loss was observed in any of the participants.

In individuals who regularly maintained oral hygiene, a certain amount of dental calculus did not cause significant attachment loss, whereas in those who did not maintain regular oral hygiene, the presence of dental calculus increased the likelihood of attachment loss by fivefold. The reported associations between dental calculus, plaque, and periodontal tissues suggest that these factors may serve as risk markers for periodontal disease.[19]

Abdellatif and Brut,[20] in their study involving 14,690 individuals aged 15 to 74, found a 20.52% association between periodontitis and poor oral hygiene. In all age groups in the study, the incidence of periodontitis was significantly higher in individuals with poor oral hygiene. Hansen et al.,[21] in their study including individuals aged 50 and older, found a 10.86% association between an OHI-S score of 2.0 or higher and pocket formation.

Restorations and prostheses may influence biofilm development and formation due to their unique chemical and physical properties. Rough surfaces contain irregular areas that are ideal for colonization and plaque formation.[22]

Smoking

When the contents of a cigarette are examined, there are over 6,000 substances that are cytotoxic, carcinogenic, and mutagenic in gaseous or particulate form. Cigarettes contain toxins such as hydrogen cyanide, ammonia, formic acid, ketones, and nitrites, and carcinogenic substances such as nickel, chromium, arsenic, benzopyrene, formaldehyde, and acetaldehyde. Each cigarette consumed contains approximately 20-30 ml of carbon monoxide and 2-3 mg of nicotine.[23]

Cigarette consumption is one of the well-known risk factors for periodontal disease. Numerous studies have demonstrated a relationship between cigarette consumption and the destruction of periodontal tissues.[24] It has been reported that smoking increases the risk of periodontal attachment or bone

loss by 2 to 8 times, depending on the stage of periodontal disease and the amount consumed.[25]

A meta-analysis including six randomized controlled trials found that severe periodontitis was three times more prevalent in smokers than in non-smokers.[26] Studies comparing smokers and non-smokers with similar plaque accumulation found that those who smoked had greater probing depth, attachment loss, bone loss, and tooth loss.[27] It has been determined that the consumption dose of smoking affects this risk and that the risk increases with increased consumption.[28]

The effect of smoking on non-surgical and phase 2 periodontal treatment: Studies have shown that individuals who smoke after undergoing non-surgical periodontal treatment, hard tissue surgery, or soft tissue surgery experience less clinical attachment gain. It has been reported that smoking delays the healing process after surgical interventions by inhibiting revascularization and suppressing growth factors.[29]

Kinane et al.[30] investigated the effects of smoking on 54 patients following antimicrobial and mechanical treatment. In all groups, they found greater attachment gain and changes in pocket depth in non-smokers.

Preber et al.,[31] in their study examining pocket depth improvement, observed lower healing in the maxillary anterior teeth of smokers following treatment. Although the exact cause is unknown, they suggested that plaque differences, greater exposure of the area to smoking compared to other regions, or a combination of these factors may be responsible.

Smoking and gingivitis: Bleeding on probing is the earliest sign of gingivitis and is used to clinically identify active disease. Smoking causes vasoconstriction in peripheral blood vessels, reducing blood flow to periodontal tissues, which results in fewer signs of gingivitis in smokers compared to non-smokers. In cases of simple gingivitis, no clinically significant difference is observed between smokers and non-smokers in terms of gum appearance. However, in studies using methods such as bleeding on probing and gingival index, it has been observed that inflammatory reactions are more prominently masked in smokers when plaque levels are high. Therefore, it has been reported that severe gingivitis is not commonly observed in smokers.[32]

Smoking and periodontitis: In clinical studies standardized for age, race, and education level, it has been reported that smokers are 3-4 times more likely to develop periodontitis and that bone loss increases in proportion to the number of cigarettes smoked per year.[33]

In a study examining 12,329 American adults aged 20 and older, it was found that current smokers were four times more likely to have periodontitis than non-smokers. The same study also reported that heavy smokers had a higher risk ratio than light smokers.[34]

Smoking and acute necrotizing ulcerative gingivitis (ANUG): Smoking is among the etiological factors of ANUG. It has been observed that smoking increases the incidence of ANUG.[35] It has been reported that nicotine-induced contraction of capillaries may cause malnutrition and decrease resistance to infection. It has been suggested that increased steroid hormone levels due to stress or reduced blood flow may suppress host defense mechanisms and exert a selective effect on dental biofilm. This situation may lead to an increase in the number of certain bacteria, such as fusospirochetes, and their high presence in areas with impaired tissue integrity.[36]

Diabetes

Diabetes is a disease caused by disorders in carbohydrate, protein, and fat metabolism, resulting from insufficient production or action of the insulin hormone.[37] Type 1 diabetes occurs due to insulin deficiency resulting from the autoimmune destruction of insulin-producing cells, while type 2 diabetes develops due to the body's resistance to insulin.[38]

The relationship between diabetes and periodontal disease has been reported in the literature since the 1960s, and periodontal disease has been suggested as the sixth major long-term complication of diabetes.[39] The relationship between diabetes and periodontal disease is bidirectional and has been defined as a two-way relationship.[40]

There is a bidirectional relationship between diabetes and periodontal disease. While periodontal infections are more common in individuals with diabetes, it has been reported that periodontal disease negatively affects glycemic control in diabetic patients.[41] There are many reasons for the increased prevalence of periodontal disease in individuals with diabetes. These include altered polymorphonuclear leukocyte function, changes in the immune response, increased oxidation, elevated LDL levels and atherosclerosis, and accumulation of advanced glycation end products in the periodontal ligament and gingiva.[42] Diabetes increases the expression of inflammatory cytokines in the periodontal tissues of individuals.[43] Increased levels of prostaglandin E2 and interleukin-1beta are observed in the gingival crevicular fluid of diabetic individuals.[44] Some studies have reported increased levels of interleukin-1beta, tumor necrosis factor, interleukin-6, interleukin-17, and interleukin-23 in the gingiva of diabetic individuals.[45] Increased expression of inflammatory cytokines leads to increased vascular permeability, inflammatory cell recruitment, reduced osteoprotegerin expression, and increased RANKL expression, thereby contributing to increased destruction.[46]

In a systematic review and meta-analysis conducted by Chavarry et al.,[47] periodontal disease prevalence and severity were found to be higher in 27 out of 49 cross-sectional studies in individuals with diabetes.

In two studies conducted on children aged 6-18 years with type 1 diabetes, the prevalence and severity of attachment loss were reported to be approximately four times higher in these patients.[48]

Some studies have found a correlation between glycated hemoglobin and pH in individuals with diabetes. Additionally, it has been reported that probing depth is greater and attachment loss is higher in individuals with uncontrolled diabetes.[49]

In their study, Nelson et al.[50] reported that the risk of new periodontal disease was 2.6 times higher in individuals with type 2 diabetes compared to healthy individuals. This study also supports the principle of causality, as diabetes developed before periodontal disease.

In a cross-sectional study conducted by Katz et al.[51] on a population of 10,590 individuals, it was reported that

individuals with high blood glucose levels had a higher likelihood of having severe periodontal conditions.

INDIVIDUAL RISK FACTORS

Individual risk factors include risk factors that are specific to the individual and cannot be modified (non-modifiable) and should be limited to this scope. Genetic factors, age, gender, socioeconomic status, and stress are examined as individual risk factors.[11]

Genetics

The predisposition to periodontal disease is based on the interaction between the microbial structure in the area and the host's genetic information. Pathogenic flora can initiate attacks that stimulate the immune response, but this is not always sufficient on its own to cause periodontal destruction. The absence of a consistent correlation between plaque quantity and disease severity, the existence of an individual dose-response curve for each person, and the fact that some individuals develop severe periodontal destruction such as aggressive periodontitis while others do not progress beyond gingivitis or early periodontitis all indicate that the host immune response is more important than bacterial etiology.[52]

There is substantial evidence that genes associated with the host immune response influence the progression of periodontal disease. Variations in different alleles lead to changes in innate immunity, antibody responses in acquired immunity, and nonspecific inflammation. This allows genetics to be considered a risk factor for periodontitis.[53]

Twin studies are conducted to investigate the effect of genetic variation on chronic periodontitis. Identical twins share the same genes because they develop in the same ovary, but fraternal twins do not have the same genetic makeup. Michalowicz et al.,[54] in a study of 110 pairs of twins aged 16 to 70, reported that monozygotic twins showed less variability in clinical attachment levels and mean periodontal pocket depths.

In another study involving 64 pairs of identical twins and 53 pairs of fraternal twins, it was reported that identical twins had a more similar predisposition to periodontitis.[55]

Numerous studies have demonstrated the role of genetics in aggressive periodontitis. Aggressive periodontitis, characterized by rapid attachment loss and severe alveolar bone destruction, is a distinct disease separate from chronic periodontitis. Unlike chronic periodontitis, it may show familial transmission or distribution. Additionally, the role of nutrition, socioeconomic status, environmental factors, and systemic pathologies has been reported in aggressive periodontitis.[56]

Genetic and chromosomal disorders associated with increased susceptibility to periodontal disease include;

- Enzyme and enzyme inhibitor defects
- Acatalasia, congenital erythropoietic porphyria, hypophosphatasia, alpha-1 antitrypsin deficiency
- Leukocyte defects
- Chédiak-Higashi syndrome, glycogen storage disease type Ib,
- Connective tissue disorders
- Ehlers-Danlos syndrome, chromosomal abnormalities of chromosomes VIII and IX
- Trisomy 21 (Down syndrome)
- Leukocyte adhesion defects types I and II, cyclic and chronic neutropenia
- Alopecia, epilepsy, mental subnormality, Papillon-Lefèvre syndrome

Gender

Studies have indicated that gender differences influence the course of many diseases. When factors such as behavioral differences, stress, hormones, and genetics are added, the prognosis of certain diseases may vary according to gender.[58]

Differences in immune responses between genders also contribute to variations in the development of periodontal diseases. Gender hormones and genes associated with the X chromosome are believed to play a key role in these mechanisms.[59]

Sex hormones (estrogen, testosterone, and progesterone) affect immunity. In general, testosterone reduces the immune response, while estrogen increases it.[60] Hormonal changes during puberty, menstruation, pregnancy, and menopause in women, as well as the use of hormonal supplements, have been associated with the development of gum inflammation.[61]

Analyses of NHANES data have estimated that men have approximately 50% higher prevalence of periodontal disease. Compared to women, men had 33% more mild, 28% more moderate, and 180% more severe periodontal disease.[62]

Studies have indicated that women visit the dentist more frequently, place greater importance on oral hygiene, and are more compliant with treatment. Additionally, the National Health and Nutrition Examination Survey conducted in the United States reported that women develop periodontal disease less frequently.[58]

Age

The relationship between periodontal disease and age remains unclear. Early studies indicated that the prevalence and severity of periodontal disease increased with age, suggesting that age could be a predictor of periodontal tissue loss.[63] However, over the years, the notion that periodontal disease is exclusively found in the elderly has been questioned. Studies have indicated that age may not increase susceptibility to the disease but rather reflect the cumulative effect of prolonged exposure to actual risk factors[64] and that periodontal disease can begin in youth/early adulthood.[65]

It has been determined that an individual's level of susceptibility to periodontal disease is more important than age and that the disease develops at an earlier age in individuals with high susceptibility. The effect of age is not the same for periodontal disease and attachment loss. While a significant relationship has been found between increasing age and clinical attachment loss, only a minimal relationship has been observed between age and periodontal disease. After adjusting for common variables such as oral hygiene levels and access to dental services, the effect of age on clinical attachment loss was found to decrease.[66]

In the 1985-1986 U.S. National Survey, where the diagnostic criterion was the presence of 4 mm or more of attachment loss

in one or more areas, the prevalence of the disease was 3.8% in the 25-34 age group and 53.6% in the 55-64 age group.[67]

Another study examining radiographs taken at 10-year intervals found that the annual average alveolar bone loss is between 0.07 and 0.14 mm for individuals aged 25-64, and 0.28 mm for those aged 70.[68]

In a study by Grbić et al.,[69] which included age, dental calculus removal, root surface smoothing, and a seven-month follow-up, a strong association was found with attachment loss of 2.5 mm or more. In the study, 35% of the 30-39 age group showed additional attachment loss, while this rate was 89% in the 60-69 age group.

Socioeconomic Status

When examining data collected in 2014 from 28 European countries, it was observed that unmet oral and dental health needs were higher among low-income groups due to income levels, geographical conditions, and limitations in the healthcare systems they were part of.[70]

Another study examined differences in access to health services between low- and high-income groups resulting from urbanization and reported that people living in rural areas had less access to oral and dental health services across all income groups.[71]

The incidence of gingivitis is higher in people with low socioeconomic status and poor oral hygiene. This situation appears to be related to the frequency of visits to the dentist and dental awareness when compared to socioeconomic status. Socioeconomic indicators are reliable predictors of periodontitis. Differences in access to resources and services that can influence preventive behaviors play a role in the socioeconomic status of periodontal disease. Studies also indicate that education has a greater effect than income in positively influencing the level of periodontal disease.[72]

It has been reported that individuals who brush their teeth twice a day and acquire this habit at an early age develop fewer cavities. The socioeconomic status of families has been reported to be effective in acquiring this habit.[73]

Bostancı et al.,[74] in a study comparing the periodontal status of students attending private and public schools, found that students attending public schools had higher CPITN values. Additionally, Gökalp et al.,[75] in a cross-sectional study involving 7,833 individuals, found that those living in rural areas had more severe periodontal problems.

In a cross-sectional study examining risk factors for periodontal disease, it was found that the prevalence of moderate or severe periodontitis was higher among individuals with lower educational levels. Additionally, the study revealed that the prevalence of moderate or severe periodontitis increased as income levels decreased.[76]

Stress

Stress is among the etiological factors of many inflammatory diseases, including periodontal diseases.[77] Stress is thought to have a negative effect on the formation of the immune response.

During stress, the sympathetic nervous system triggers the release of norepinephrine, which suppresses the immune response.[78] Stress increases the production of glucocorticoid hormones from the adrenal cortex and the synthesis of corticotropin from the pituitary gland; it also reduces the synthesis of pro-inflammatory cytokines.[79]

Hilgert et al.[80] demonstrated that elevated cortisol levels in high-stress conditions are associated with higher levels of periodontal disease.

A study involving 1.426 individuals showed that those who demonstrated excellent coping skills in the face of similar economic difficulties had a lower risk of periodontal disease.[81]

In a small study of 23 individuals examining occupational stress and the progression of periodontal disease, it was reported that increasing age, low socioeconomic status, low job satisfaction, and aggressive behavior significantly increased attachment loss.[82]

Studies have shown that saliva flow rate decreases under stress, and emotional stress affects saliva pH, leading to the development of gum diseases.[83]

The increased incidence of necrotizing ulcerative gingivitis under emotional and physiological stress suggests a connection between the two conditions. Emotional stress affects hormone levels in the bloodstream, which in turn leads to changes in the immune response. It has been reported that the prevalence of periodontal disease increases in stressful situations such as the loss of a close relative, divorce, and economic difficulties.[84]

Stress also affects behavior. In stressful situations, increased smoking, poor oral hygiene, decreased visits to the dentist, and changes in eating habits may be observed. These behaviors can have a negative effect on the periodontal disease process and cause a decrease in the immune response.[85]

RISK INDICATORS FOR PERIODONTAL DISEASE

Periodontal disease risk indicators are risk factors that have been identified in intergroup studies but have not been confirmed by long-term research. These indicators are risk factors that may cause disease or are associated with disease. HIV/AIDS, osteoporosis, and irregular dental checkups fall into this group.[11]

HIV/AIDS

Acquired immune deficiency syndrome (AIDS) is the most severe clinical manifestation of human immunodeficiency virus (HIV) infection. AIDS was first defined in 1981 in the United States as a viral infection that can affect all body systems, has no effective treatment, and can be fatal. AIDS is more common in intravenous drug users, hemophiliacs, patients requiring blood transfusions, bisexual and homosexual men, heterosexual women, and their sexual partners.[86]

In most patients, the first symptom of HIV infection may be oral lesions caused by opportunistic fungal and viral agents. Due to the suppression of the immune system, oral candidiasis is the most common oral condition observed in individuals with AIDS. Additionally, hairy leukoplakia, Kaposi's sarcoma, ulcers, herpes simplex, papilloma, and periodontal diseases may also occur.[87]

More severe necrotizing periodontal diseases often develop in individuals with AIDS. These are caused by spirochetes, fusiform bacilli, gram-negative bacteria, and other microorganisms, as well as malnutrition and poor oral hygiene.

It begins with necrosis of the dental papillae, followed by pain, bleeding, foul breath, fever, and lymphadenopathy. The formation of band-like erythema on the gingival margins by Candida albicans in AIDS patients is defined as marginal gingivitis.[88] HIV infection and AIDS are considered risk factors for periodontal disease.

The prevalence of necrotizing ulcerative periodontitis in AIDS patients and HIV-infected individuals is not precisely known. Some studies indicate that the prevalence of periodontal disease is higher in these individuals compared to others with similar oral hygiene, with prevalence rates ranging between 4% and 5%.[89]

In a prospective study, 114 homosexual and bisexual men were examined over a period of 20 months, and the incidence of active periodontal disease was found to be 6.16 times higher in individuals with CD4 cell counts below 200.[90]

In the first studies conducted on AIDS patients, the prevalence and severity of periodontal disease were higher. However, subsequent studies reported less severe periodontal symptoms. This finding is thought to be related to the successful suppression of the immune system in HIV-positive individuals through high-activity antiretroviral therapy and the continuous development of other medical agents.[91]

Robinson et al.[92] found no difference in the progression of periodontal disease between HIV-positive and HIV-negative individuals in a 12-month follow-up study.

Hofer et al.[93] demonstrated that HIV-positive individuals can be successfully protected in a manner similar to uninfected patients. The specific IgG subclass responses to periodontal pathogens were found to be alike in both HIV-infected and uninfected individuals.

Osteoporosis

Osteoporosis is a metabolic skeletal disease characterized by an increase in bone fragility and fracture risk, as well as a decrease in mineral density, due to deterioration in the microarchitecture of bone tissue.[95]

Non-modifiable risk factors for osteoporosis include age, female gender, family history, previous fractures, race, ethnic origin, menopause, hysterectomy, long-term glucocorticoid therapy, rheumatoid arthritis, and primary/secondary hypogonadism in men. Modifiable risk factors for osteoporosis include inadequate calcium and vitamin D intake, insufficient consumption of fruits and vegetables, excessive protein, sodium, and caffeine intake, eating disorders, insufficient physical activity, frequent falls, and tobacco and alcohol use.[96]

In short-term cross-sectional studies, where most participants were postmenopausal women, it has been reported that women with low bone mineral density had a higher incidence of gum recession, gum inflammation, and attachment loss (von Wowern et al., 1994; Mohammad et al., 1996, 1997; Tezal et al., 2000). However, some studies have not reported such an association (Weyant et al., 1999; Lundström et al., 2001).[97]

Payne et al.[98] reported greater alveolar bone loss in osteoporotic women in their long-term studies. However, Reinhardt et al.[99] reported that serum estradiol levels had no significant effect on attachment loss in their 2-year follow-up studies (**Table**).

Irregular Dental Checks

The issue of whether irregular dental visits are a risk factor for periodontal disease is controversial. One study showed an increased risk of severe periodontitis in patients who did not visit the dentist for three years or more, while another study found no significant difference in attachment and bone loss between those who received dental treatment and those who did not over a six-year period.[11]

Altındal et al.[101] conducted a study between 2016 and 2018 with 1,758 participants, finding a statistically significant association between the diagnosis of periodontal disease and the frequency of dental visits. According to the study results, the majority of healthy individuals reported having previously visited a dentist, while the majority of those diagnosed with periodontitis and gingivitis stated that they had never visited a dentist before.

RISK PREDICTORS/MARKERS FOR THE FUTURE

Signs of future risk prediction/risk signs are associated with an increased risk of disease but do not cause disease. These factors have been identified in intergroup and long-term studies. In this context, factors such as gingival recession and a history of periodontal disease can be examined.[11]

Bleeding During Probing

Dentists associate bleeding during probing with periodontal areas at high risk of future deterioration and use the finding of bleeding during probing as an indication for treatment. However, recent studies have shown that this use should be questioned.[102]

Table. Studies evaluating the relationship between systemic bone loss and periodontitis[100]

Author	Periodontal measurement	Systemic bone measurement	Correlation
Elders et al.	Alveolar bone height	Dual photon absorptiometry, L2-L4, metacarpal thickness	No
Klemetti et al.	Index of periodontal treatment needs in the community	Dual-energy X-Ray absorptiometry of the spine and hip	No
Hildebolt et al.	Clinical detachment	Dual-energy X-Ray absorptiometry of the spine and hip	No
Mohammad et al.	Clinical detachment	Dual-energy X-Ray absorptiometry of the spine and hip	Yes
Lundstrom et al.	Alveolar crest height	Dual-energy X-Ray absorptiometry of the hip	No
Mohammad et al.	Clinical detachment	Dual-energy X-Ray absorptiometry of the spine	Yes
Taguchi et al.	Alveolar crest height	Dual-energy X-Ray absorptiometry of the spine and femoral neck	No
Ishii et al.	Alveolar crest height	Dual-energy X-Ray absorptiometry of the femoral neck	No
Brennan et al.	Clinical detachment	Dual-energy X-Ray absorptiometry of the spine, hip, and forearm	Yes
Payne et al.	Alveolar crest height	Dual-energy X-Ray absorptiometry	Yes
Yoshihara et al.	Clinical detachment	Dual-energy X-Ray absorptiometry	No

Gbric and Lamster,[103] in their 6-month study of 75 patients, found that 68% of areas showing active disease had bleeding, and 57% of areas without active disease also had bleeding.

Kaldahl et al.[104] conducted a study to determine active disease areas, examining patients who had undergone maintenance therapy for two years. Their findings concluded that the presence of bleeding during probing does not predict future attachment loss.

Previous Periodontal Disease

Many clinical studies have shown that individuals who have had periodontal disease in the past are at higher risk of developing the disease again in the future compared to those who have not had it.[105] The most common situations in which disease recurrence occurs after treatment include poor oral hygiene, smoking, systemic diseases such as diabetes, and non-compliance with treatment.[106] Adherence to regular supportive periodontal therapy (SPT) plays an important role in reducing disease recurrence and tooth loss.[107]

In a meta-analysis conducted to evaluate the effectiveness of different methods in assessing disease recurrence and managing recurrent disease in individuals who had undergone long-term supportive periodontal care (SPC) and had previously received periodontitis treatment, the rate of patients experiencing multiple 2 mm attachment loss was found to be 24.8%.[108]

In a study conducted in Iran involving 50 patients (29 men and 21 women) who had completed periodontal treatment but did not adhere to the maintenance phase, a significant association was found between the initial diagnosis and the recurrence rate of periodontitis (p=0.017). Additionally, a significant relationship was found between the number of years post-treatment and periodontitis recurrence (p=0.027, r=0.353).[109]

ETHICAL DECLARATIONS

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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